values were greater than the respective median. The difference in treatment effect on baseline disease severity sub-group was analyzed using analysis of covariance (ANCOVA).
Results: In total, 3235 patients comprised the ITT population of the pooled efficacy analysis. Out of these, 3214 patients were included in the disease severity analysis [low (n = 923), medium (n =1456) and high (n=835)]. Mean OA pain at baseline was 79 mm in the high disease severity, 65 mm in the medium disease severity and 53 mm in the low disease severity subgroup. The greatest reduction in OA pain intensity with lumiracoxib and celecoxib compared to placebo, was seen in the highest disease severity group (lumiracoxib [-9.30mm; $p \leq 0.001$] celecoxib [-6.70 mm; p=0.004]) as compared to the medium disease severity group (lumiracoxib [-5.48; $p \leq 0.001$], celecoxib [-4.76; p=0.006]) and low disease severity group (lumiracoxib [-4.74 mm; p=0.012] celecoxib [-4.74 mm; p=0.030]). Similar results were seen for patient's global assessment of disease activity and the functional status, assessed by WOMAC™ LK3.1 total score (Table 1).
Conclusions: Lumiracoxib provides effective pain relief and improvement of symptoms in all subgroups of OA patients defined by disease severity at baseline. However, there is a trend for greatest efficacy in the subgroups of patients with highest disease severity.

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IS SELF-REPORTED IMPROVEMENT IN OSTEOARTHRITIS PAIN AND DISABILITY REFLECTED IN OBJECTIVE MEASURES?

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Purpose: Osteoarthritis (OA) is perceived as a major public health problem, and today, various clinical outcome methods are used to see the improvement of the management for this condition. The purpose of this study was to determine if self-reported improvements in pain and function correlate with improvement in objective measures of disease in OA.
Methods: Eighty female patients (mean age: 52.3 ± 8.7 years) with bilateral OA of the knee were assessed by some tests including [sociodemographics, body mass index (28.34± 4.88 kg/m^2, and a self-reported questionnaire-Western Ontario and McMaster University Osteoarthritis Index (WOMAC) scores] and physical [muscular strength by ISOMED 2000 isokinetic dynamometry, proprioception by determining deficit at the knee joint using by Functional Squat System, Timed Get up and Go (TGG) for walking duration, visual analog scale for measuring the intensity of the present pain, warmth, effusion] and clinical examination of the knees (Altman Grade II). Subjects received closed kinetic chain in clinic and home-based structured exercise program 5 days a week for 4 weeks, totally 20 sessions. All tests were done before and after the 4-week treatment period.
Results: There were no significant correlation between pain at rest and night, during activity and proprioception deficit in left (r= 0.05, r= -0.16, and r= -0.14; $p > 0.05$) and right (r=0.22, r=0.07, and r=0.19; $p > 0.05$) knee joint pre-treatment, respectively. Besides; there were found a correlation after treatment between pain at rest and night, with activity and proprioception deficit in left (r= 0.32, r= -0.37, and r= -0.29; $p < 0.05$) and right (r=0.53, r=0.39, and r=0.2; $p < 0.05$) knee. The peak torque of isokinetic muscular strength and proprioception of participants were improved after rehabilitation. Of eighty eligible patients, WOMAC scores were improved after immediate treatment in clinical-based exercise group, 46.4% and 56.4% reported the improvement in pain and function, respectively. However, improvements in WOMAC scores were not associated with improvements in any of the other measures (Left knee peak torque (PT), r= -0.28 and right knee PT, r= -0.29; $p > 0.05$ for all). There was a significant correlation between proprioception scores and TGG test either before (r= -0.31; p=0.016) ($p < 0.05$) or after (r= -0.4141; p=0.001) ($p < 0.01$) rehabilitation. The findings at 4th weeks after enrollment were that the quadriceps strength was slightly increased but the walking duration was decreased when compared with the initial assessment. An exercise frequency of 20 sessions in a month was sufficient to improve muscle strength and walking distance.
Conclusions: Seventy-three of participants reported significant improvements in WOMAC pain and disability after treatment. Both closed kinetic chain and home-based exercise program produce a significant improvement in strength and walking ability, especially in the first months. In the assessment of knee OA, muscle strength, proprioception, and walking ability can be used to measure the effect of improvement of pain and disability after treatment programs.

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DOES INCREASING OBESITY INCREASE THE PROGRESSION OF KNEE OSTEOARTHRITIS?

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Purpose: Results of studies that have evaluated obesity as a risk factor for the progression of structural damage in knees with osteoarthritis (OA) are conflicting. However, researchers who design clinical trials for structure-modifying drugs for OA (SMOADs) often consider "weighting" the study cohort with patients with a higher body mass index (BMI), in the hope of enriching for patients who will have more rapidly progressive OA. We asked whether, among obese patients with knee OA, a higher BMI was associated with an increased rate of joint space narrowing (JSN).
Methods: In 60 women with symptomatic OA (KLG2 or 3 in a standing AP radiograph) and a mean BMI (± SD) of 37.2 ± 5.1, medial tibiofemoral JSN over 12 months were measured in modified Lyon schuss (mLS) radiographs. We compared these findings to those in 81 age-matched, non-obese, asymptomatic, KLG0 women (BMI = 23.5 ± 2.3).
Results: Among the OA patients, a higher BMI tended to be associated with a higher KLG. Mean BMI was 36.0 ± 4.5 in KLG2 patients and 38.4 ± 5.4 in KLG3 patients (p=0.06). Mean JSN in the 81 controls was 0.02 ± 0.25 mm (p=0.51), whereas in the 30 KLG2 knees and 30 KLG3 knees, mean JSN was 0.12 ± 0.31 mm (p=0.08 vs. KLG0) and 0.32 ± 0.50 mm ($p < 0.0001$ vs. KLG0), respectively. KLG3 knees were more homogeneous with respect to JSN than KLG2 knees (SRM = 0.64 vs. 0.39). BMI was not related to JSN in OA knees.
Conclusions: Among these OA patients, with a BMI $\geq$ 30, there was no evidence that progressively higher BMIs were accompanied by a progressively increasing rate of JSN. We will determine whether a longer period of observation will yield different results. In the mLS views, 12 months was sufficient to detect JSN in KLG2/3 knees, relative to knees of nonarthritic controls (p=0.0001). Because none of our OA patients had a BMI <30, this study cannot not address the question whether JSN is more rapid in overweight or obese subjects than in knee OA patients of normal weight. For an SMOAD trial, these data suggest that recruitment of patients with a BMI much beyond 30 will not enrich the sample in subjects who will exhibit more rapid JSN than would patients with a BMI of 30.